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UNIVERSITY OF TORONTO MEDICAL JOURNAL

Volume XIV—1936-37

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THE UNIVERSITY OF TORONTO MEDICAL JOURNAL

(An Undergraduate Publication)

This Journal is published six times a year, November to April, by the Medical Society of the University of Toronto, to serve as a medium for student expression and to bring to the student recent articles of scientific interest.

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Vol. XIV Toronto, November, 1936 No. 1

It is with pleasure that the Editorial Staff present to students and graduates the first issue of Vol. XIV. of their journal. Although this publication is now to appear under a cover of newer design, we realize that its worth lies in the pages of more modest tone which carry its contents.

The student support given to recent changes in the constitution of the Medical Society, which affect this paper particularly, assure us of their sincere interest in the Journal. But such interest will bear its choicest fruits in the articles they are invited to contribute. Individuals have from time to time criticized the custom of undergraduate publication. However, this comes from those who have failed to derive the full benefit of the cause, which can come only to contributors as the result of energies expended in preparing a paper containing accurate and well-arranged scientific information.

A word on policy may be in order here. It is hoped that throughout the year there may be presented in this Journal a series of articles dealing with topics which affect students, practitioners and patients. We refer to "Educational Methods" and "Health

Insurance," ("State Medicine" or "Socialized Medicine.")

As students, we all have had mingled feelings of satisfaction and dissatisfaction in the medical course as presented to us. There is no doubt that the quality of graduate from this faculty and university ranks high among the profession throughout the world. Although changes may little affect the ultimate result, the formulation of improved paths to the goal is part and parcel of a progressive institution. We feel the Staff shares this sentiment keenly and appreciate our interest in the matter. Accordingly it is appropriate that our newly appointed Dean, Professor Gallie, should favour the first issue with his pen.

A new system of dispensing medical assistance to the sick would come as a great relief to many physicians and patients throughout the country. Only by an extensive study of this problem will the most satisfactory arrangement result. We who are students now will find ourselves on the crest of the wave of change which is to come. Is it not, then, appropriate that we fit ourselves to contribute to the problem? Mr. Kerr's article presents the trend of thought in vogue to-day upon this topic.

In order that students may comment upon these topics, the Editor would be pleased to receive letters, brief and to the point, for publication. Letters must be signed by the writer to insure us of authenticity of source, but names will be withheld from publication unless otherwise requested.

To the Freshmen.

We heartily welcome the new class of '42. Long is the course to be followed, and tedious the hours with tiring eyes before the goal is gained, but many will be the bright spots upon the way and great the satisfaction when the end is reached. Regardless of the topic, the fact that interest is aroused is the finest index that some familiarity has been established with the subject studied. Those of you who may find study a bore can change your disinterest to delight by considering each subject a sector of the circle which may be considerd as representing an understanding of medicine. Only the years of experience can enlarge it.

We can do little more than wish you brilliant success and satisfaction in your undergraduate days.

The Deanship.

It is with regret that we heard of Professor FitzGerald's resignation, after more than four and one-half years of earnest service. For his continued brilliant accomplishments and happiness, may we express the good wishes of the student body.

We are fortunate to have as successor Dr. W. E. Gallie, Professor of Surgery. In the name of the student body, we take this opportunity to welcome Dean Gallie and to offer sincere co-operation during the following years.

The Cover Design.

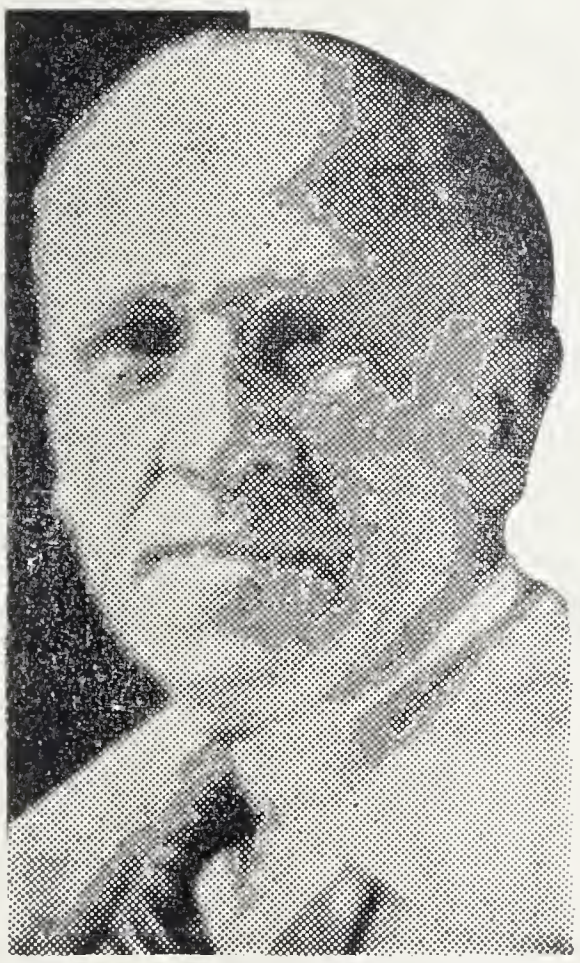
The new cover was designed by Irving Warren of '37, and submitted as an entry in the contest sponsored by the Medical Society.

We congratulate Mr. Warren on his artistic concept and skill and thank other contributors for the interest and work they exhibited.

In Memoriam

Professor Oskar Klotz, M.B., M.D.C.M. (McG.), F.R.S.C. (C.),
Late Professor of Pathology and Bacteriology,
University of Toronto.

These lines are sacred to the memory of Professor Klotz, who passed away at his home early on November 3, 1936, and are prepared as a token of the high esteem in which he was held by those who knew him.



Professor Klotz was born in Preston, Ontario, January 21, 1878. He graduated from the University of Toronto as M.B., in 1902, and as M. D. C. M. from McGill in 1906. He did extensive post-graduate work in Montreal, New York and Germany, following which he held the chair of Pathology and Bacteriology at the University of Pittsburgh, Faculdade de Medicina at Sao Paulo, Brazil, and at this university since 1923. His researches in yellow fever, arterial disease and cancer brought to him and this university world renown and prestige.

His untiring energies carried him on in active work until within a few brief days of his end, in spite of indifferent health over a long period.

That which raised him so high in the esteem of students was the authority they realized in the man, the completeness with which he knew and presented his subject, not only as facts but in a setting possible only by one who was well-read, a deep thinker and a friend of all life. His bearing in learned gatherings, so superior to that of others present, the gentlemanly and authoritative manner in which he discussed with the unlearned, topics covering many fields, betoken the splendid man and scholar he was. In one's undergraduate days one thrilled that such a world-renowned figure was so approachable.

It was with the deepest sorrow that we heard of his departure, for he left a gap too wide for any single man to fill, and we lost a delightful man and warm-hearted friend.

SOME few years ago in Barrie an important event occurred. While there have probably been lots of fairly important events occurring in Barrie, still this one was of particular importance to Toronto, as it had a very definite bearing on your University training. The event was the birth of William Edward Gallie, as he was subsequently christened.

W. E. Gallie, M.D., F.R.C.S. (Eng.), F.R.C.S. (C.),
Dean of the Faculty of Medicine, University of Toronto.
—Photo by George Freeland.

His early life was spent among the rigors of the northern climate, the intense cold of its winters and the heat of its summers; the sports in winter being hockey and skating, not fancy, and in summer, lacrosse and swimming. The boy became adept at these pastimes. I never heard him complain of any difficulties he had in preparing himself for or in passing the examinations that he took as a matter of course, but there were some trials that entered his young life, and these were mostly in the form of manual labour. He was the grief of his father, who was unable to get him to do what he considered useful work. The lad apparently was bound for a life of utter uselessness, as he showed no desire to undertake reasonable work which would allow him to earn his living in an ordinary way.

It is very much a matter of doubt as to whether or not he ultimately decided upon his professional life as the lesser of two evils, nor is the writer aware of the motives which led him into the professional life. It is not likely that he went into it from an absolutely altruistic motive such as a pure unadulterated wish to be of service to suffering humanity, but rather from another cause which would make one believe that he had a consuming urge against which he was entirely powerless, to follow out some idea he had developed in his mind of proceeding to a surgical career. There are many incidents and periods in his life which those who know him well would recognize as the irresistible which press him on toward a goal which he has visualized. It is probable that as a little lad he was very ambitious and would spare no effort to accomplish the task to which he had set himself. Whether it were long distance or endurance swimming, lacrosse or hockey, he would devote to it every bit of his time and energy until he had acquired the proficiency in it at which he aimed.

He came to the University as a very young student and entered the Faculty of Medicine. Entering at the same time in his class were several men who were brilliant then and who have since made great contributions to both medicine as a whole and the University of Toronto in particular. This year has already given two Deans to the Faculty of Medicine. Dr. Gallie during his time at the University of Toronto played hockey by choice with his home team. The reason for his doing this is too long a story to consider here, but it was his choice. His course at the University was taken in his stride, and during that course he had an opportunity to observe and meet the men who subse-

quently had so much to do with molding his plastic mind into definite channels. The teachers who were famous in those days were: Dr. Primrose in Anatomy, Dr. George Peters in Surgery, and a man who had a tremendous influence on Dr. Gallie, Dr. C. L. Starr, who was practising orthopaedic surgery along with his general surgical practice and who was working at the Hospital for Sick Children. With three such brilliant preceptors it was not to be wondered at that a character such as his would be set aflame by the possibilities of surgery and of life as a surgeon.

Following graduation he spent some time as a rotating intern at the Hospital for Sick Children and at the Toronto General Hospital. Following this he got an internship in the Ruptured and Crippled Hospital in New York, which then numbered among its staff such men as Royal Whitman, Coley, and the Gibmoys.

Following his return to Toronto he became attached to the General Hospital and became assistant to the late C. L. Starr. In 1912 the latter became Surgeon-in-Chief of the Hospital for Sick Children, and Dr. Gallie was his assistant there. He kept up the enthusiasm for golf acquired in New York, and for the next two or three years his energies were equally divided between his academic interest in surgery and his practical interest in golf. He needed private patients as a means of livelihood, but he was known at this time to slip out the back door of his office to escape a patient in order that he might not be late for teeing off.

About this period he conceived the idea of taking his English Fellowship. In those days it was necessary to go to London to try the Primary. He went blithely off to cross the ocean. He sailed from Philadelphia on a slow boat. This was a very sad experience. He had no idea that the ocean was such a horrid place. He was still living when they landed and was taken off the boat. In a day or so he recovered sufficiently to try his Primary Fellowship, which he succeeded in getting. Later on he completed the Fellowship during his stay in England with the Canadian Expeditionary Force.

Hockey again claimed some of his attention and for a few years he became the coach of the Varsity team. Some years later while he was Presiden of the University of Toronto Hockey Club, the team he had coached grew to maturity and as the University Grads, won the World's Amateur Championship at the Olympics.

During these years his interest, having become more centred in surgery, and as his responsibility increased at the hospital, as he read more papers and spoke at more meetings, his practice grew to a degree where he had to relinquish his golf. The wheel having started to spin, gained momentum, and presently he found himself so immersed in surgery, both as to practice and academic duties, that he had no time for anything else. In 1923 he became Surgeon-in-Chief of the Hospital for Sick Children, and in 1929 succeeded his old preceptor, Professor Starr, to the Chair of Surgery and the position of Surgeon-in-Chief of the Toronto General Hospital. In this atmosphere he is now saturated.

Twenty-two years ago he was married. This undoubtedly was the best thing he ever did for himself. It is not too much to say that his present interests are centred upon youth, both in his own family and in the profession, particularly in the young men that may be brought along in the University who will be able to bring honour and prestige to the profession of medicine and the institutions to which they are attached. His duties and responsibilities have multiplied year by year until at the present time he is one of the busiest men in the whole of Canada. It is very amusing to think of his early days when his father had doubts about this boy's ability to work, and I think he is still a great mystery to his father, but of course to his mother he always was wonderful.

D. E. ROBERTSON, M.D.

IDEALS OF THE TEACHING OF SURGERY

By W. E. GALLIE,
Professor of Surgery.

With all the experience we have had in the teaching of Medicine and Surgery, it would seem that a nearly perfect system could easily be devised and, with whatever minor variations were necessary to make it fit into local conditions, adopted in all Class A Medical Schools. Unfortunately, however, no unanimity exists as to what are the basic requirements of a satisfactory course and in consequence there are almost as many curricula as there are schools.

The most noticeable difference in the plan of surgical teaching in various Medical Schools is in the amount of actual practice that the student is given in the diagnosis and treatment of surgical diseases. Twenty years ago the course in Surgery in this School consisted solely of lectures and ward clinics. The student wrote only a few histories and took no part whatever in the treatment. This is still the plan of teaching in many schools and is based upon the idea that training in actual practice should be postponed to postgraduate years. In these schools apprenticeship does not commence till after graduation.

This plan has two grave defects. In the first place it fails to give adequate training to that part of our students who are unable to supplement their Medical Course with internships and who must go directly into general practice. These men have been notorious for their ignorance both of diagnosis and treatment and for their utter unpreparedness to deal with the simple minor surgical emergencies met in the daily round of practice. In the second place, it fails entirely to command the interest of the student. Nothing so thoroughly destroys an enthusiasm for clinical study as lectures, text-books and clinics in the absence of opportunity to come in close and frequent contact with patients. I recall the thrill of pleasure with which I discovered as an intern that the practice of surgery was not the dreadful bore that my teachers had led me to suppose it to be.

Some years ago these defects in our system, which applied to Medicine and Obstetrics almost as much as to Surgery, were fully recognized by the Faculty and an attempt made to remedy them by adding the Sixth or clinical year. It was intended that this should provide for the student an apprenticeship which would help him to bridge the gap between school and practice. This clinical year has undoubtedly improved matters, but in the case of Surgery at least, the improvement has not been, until recently, sufficient. The trouble has been that the six or seven students allotted to each surgical division for a trimester of ten weeks seldom seemed to get down to work and they spent most of their time waiting around the corridors for somebody to tell them something, instead of joining the interns and nurses in the daily work of the wards.

To get over this difficulty we have made what is a fundamental change in the routine of the hospital and have made the students attached to each surgical division a part of the ward organization and in reality externs of the hospital. These students now take all the histories and make and record all the physical examinations and are as much a part of the ward organization as is the junior intern to whom they are directly responsible. The result has been a tremendous improvement in the interest the students have taken in their work and in the amount of the surgical knowledge they have acquired.

In order that this radical change in the ward routine may work smoothly it is necessary that students be avail-

able the whole year round and it was because of this that volunteers were called for last June to serve during the summer months. This experiment proved so successful that it is our intention to continue it each summer and to enlarge it to include as many departments as are likely to benefit from it.

The striking result of the summer experiment was that at the clinical examinations held in September, these students gave evidence of a better practical knowledge of Surgery than any similar group of students had ever done before. Each had written one hundred histories and done all the work necessary to prepare the cases and had taken part in the operation and after treatment. They had, in fact, had as good a training as most rotating interns. And now that the regular term has opened, opportunity is offered to those students who took their surgery in the summer to receive special training during this trimester that they would otherwise have devoted to surgery. Thus one man is attached to the Hospital for Sick Children, one to the Urological Service at St. Michael's, one to the cancer clinic in the Dunlap Building with Dr. Richards and one to the Neuro-Surgical service at the General. These students are receiving a surgical training such as no students in this School ever received before.

Our enthusiasm for the changes that have been made should not be interpreted as meaning that we have a lessened regard for post-graduate work in the form of internships. We believe that if it is at all possible every graduate should spend at least one year as an intern. Its real meaning is that we are convinced that the development of this system of apprenticeship in the final year has greatly increased the interest of the student in his work and has resulted in his acquiring a much better theoretical as well as practical knowledge of the diagnosis and treatment of surgical disease.

The success of the summer experiment in the teaching of surgery has been so striking that we propose to continue it each year. Towards the Spring applications will be received and eighteen or twenty students will be selected. Preference will be given, other things being equal, to the students of the highest standing. Care will be taken to provide for these students special opportuntiies for such work as they desire during the regular trimester in which they would ordinarily be taking their surgery and good work done during the summer will be remembered when interns are being selected.

Coramine as a Cardio-Tonic.

To decide the question whether Coramine has an effect on the heart, this substance was tested on Starling's cardio-pulmonary preparation. The results were: On the insufficient heart, Coramine increases diastole, reduces the venous pressure, thereby diminishing the venous congestion; it increases arterial pressure and the minute volume. The increase of the minute volume improves the general circulation. These results correspond to the actual clinical results and justify the use of Coramine in cardiac insufficiency.

—Abstract.

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VARICOSE VEINS AND BLOOD STASIS

By JOHN HAROLD COUCH, M.B., F.R.C.S. (Edin.), F.R.C.S. (C.),
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Difficulties in maintaining the long up-hill flow of blood from the foot to the heart are inevitable, as a result of man's election to walk in the upright position, so that his hands may be left free to do things.

Any failure in the efficiency of the machinery for maintaining this up-hill circulation results in stasis of fluids at the bottom of the system.

Clinically, stasis manifests itself in two forms—

(1) Stasis of blood in veins—or varicose veins.
(2) Stasis of serum, lymph, or tissue fluid in tissue spaces—lymphoedema.

These two independent and yet related forms of stasis will be further discussed under separate headings.

Venous Circulation of Leg.

The varicose veins of everyday practice manifest themselves as tortuosities of the long saphenous veins in the legs. The short saphenous vein is less often involved, for reasons which become clear as we discuss the processes which result in such dilatation.

The venous circulation in the legs is divided into two groups. The superficial venous return consists of the long and short saphenous veins. The long saphenous vein is formed on the dorsum of the foot or just in front of the medial malleolus, and courses almost straight up to the saphenous opening, where it ends by pouring into the femoral vein. Throughout this course, it lies in fat between the skin and deep fascia, and is unsupported by any strong structure, such as muscle or deep fascia. The function of the superficial venous system is to drain the skin, fat, and superficial tissues of the foot and leg. It is this superficial set of veins that is so prone to varicosities.

The deep venous system comprising the femoral and its tributaries lies under deep fascia, well supported by strong muscles, the massaging action of which help in emptying the whole system. The function of the deep system is to drain muscles, bones, joints and all structures beneath deep fascia. These veins never become varicosed in the usual sense of the word.

The Stasis Factor.

The unsightly appearance of varicose veins is not the only reason why we are concerned with their cure. Stasis, which is a constant and inevitable accompaniment of varicose veins, is the real cause of all the troubles they produce. Sometimes, in admiring the large tortuous "bunches of grapes," we lose sight of a more insidious mischief going on as a result of their presence.

It matters not by what manner of channel blood reaches tissues, so long as sufficient good blood is provided for the nourishment of the parts. Without free exchange of blood, however, vitality of tissue is quickly impaired. And that is exactly the situation which obtains in such patients. There is neither sufficiently rapid exchange of blood, nor is the blood of ordinarily good quality for satisfactory tissue nourishment. Blood in varicose veins is almost stagnant, the flow is extremely slow, and even in the reverse direction. A slow, reversed flow is the essential characteristic which differentiates circulation in varicose veins from that in useful veins, whether straight or tortuous. The stasis of blood in big, cavernous puddles is responsible for all the consequences, both small and great, of varicose veins. It is, therefore, from the point of view of blood stasis that we discuss the problem.

Causes.

The well-recognized picture of varicose veins is not produced by any one

cause, but rather by a combination of several factors acting together.

1. *Heredity:* Over 50% of varicose-vein patients give a positive family history of varicose veins. Less than 50% of the general population give such a family history. Therefore, there must be a positive co-efficient of co-relation between the occurrence of relatives with varicosities and varicosities appearing in any one individual.

2. *Weak Walls:* Microscopic examination actually reveals that the veins inherited by these patients, that is to say, varicose veins, and veins not yet varicose, but from the same individual, have thinner walls, with less supporting structure than the normal.

3. *Garters:* Tight circular garters may conceivably interfere to some extent with the easy return of blood in the long saphenous vein as it lies close under the skin. We have not been impressed with garters as an important cause, but Stuart McAusland feels very strongly on this point, and says that they are an evil against which one must wage unceasing war.

4. *Illness:* Veins which may already be below par cannot but share in the loss of tone which follows any debilitating illness, and are, therefore, at such time more prone to dilatation and stretching if subjected to any undue stress.

5. *Occupation:* Work which necessitates standing for long periods of time is not unlikely to result in the appearance of varicose veins. Standing still is worse than walking. Shop girls, policemen, nurses, housewives, show a higher incidence of large veins than those whose mode of life provides ample leisure, or well-regulated physical exercise.

6. *Intra-pelvic pressure:* Any tumour which presses on the iliac vessels renders more difficult the onward flow from the veins of the legs to the vena cava. The commonest intra-pelvic tumour is the pregnant uterus. Slight interference with the ready flow of blood prolonged over the whole nine months, and perhaps repeated from time to time, precipitates varicose veins or aggravates those already present.

Pathological Process.

Once a slight dilatation has been brought about, a vicious circle is established. The valve cusps fail to meet across the dilated lumen. Blood leaks back past them. Since there are normally no valves in the abdominal and thoracic veins, we have established an unbroken column of blood, extending from the foot to the level of the heart. This means a very considerable lift straight up against gravity. The lift must be accomplished by two sets of forces only.

1. *Vis-a-tergo:* The onward pressure in venous capillaries is almost nil.

2. *The sucking action of the chest in inspiration:* Negative pressure thus established is a much more valuable force than pressure from the rear in emptying of veins, both normal and varicosed. It is, however, intermittent and irregular, and in itself, is not sufficient to maintain efficient onward flow. Furthermore, any forced expiration, such as laughing, coughing, sneezing, blowing, results, since valves are wide open, in pushing blood back down into the feeble venous capillaries. The pressure in a varicose vein has been shown to be equal to that of a column of water of approximately the same height. It is much greater than the pressure in normal veins and any increase in intra-thoracic pressure is transmitted immediately to the varicosed veins, so that at times the pressure therein is greater than even arterial blood pressure. Thus is explained the difference in haemorrhage from a varicose vein and from a normal vein. Blood from a pin-sized hole in a varicosity squirts to the walls, and more than one patient has exsanguinated himself from a small hole because no one nearby had enough presence of mind to put a finger on the opening.

Results of Pathological Progress.

It is no wonder, then, that the system ultimately breaks down. The onward flow is halted and then re-

versed. Blood settles heavily back into the unsupported superficial veins. They dilate and stretch becoming huge tortuous caverns. The stream slowly reverses, and travels sluggishly downhill, damning back the inflow of fresh nourishing blood, which otherwise would enter through the arteriolar capillaries. Thus the tissues at the bottom of the column are perfused with useless stagnant blood. After a time, the blood in varicose veins finds its way along the path of least resistance, through the communicating vessels that penetrate the deep fascia, and enters the deep venous system. The deep veins, lying in strong muscles, under a tight, deep fascia, are, fortunately, able to take on and do, not only their own work, but also the work of the superficial veins.

Finally, to complete the picture, the blood goes up the femoral vein to the saphenous opening, where some of it overflows into the saphenous vein and starts downward again on its vicious circle, perfusing the tissues again with blood even more depleted than ever.

Consequences of Stasis.

The tissue which suffers most from this reverse perfusion is the skin in the region of the medial malleolus. It lies at the bottom of the stagnant column. The foot beyond suffers less, because there the veins are horizontal, communication between deep veins and superficial in the foot is more efficient, and the foot is usually supported and protected by a firmly laced shoe or boot.

The skin over and above the medial malleolus is particularly poorly equipped to face any such hardship. Nature, in an economy of space and for the sake of safety, as well as to the joy of the students in anatomy, often packs artery, vein, and nerve closely together. Again and again throughout the body we find these three, artery, vein, and nerve, running in approximately the same channels, and certainly corresponding more or less closely in their arrangements and distribution. In the superficial tissues on the medial side of the leg, we have a long saphenous vein and a long saphenous nerve. There is *no* saphenous artery. It is represented in man by a small vestigial branch running down a short distance from the anastomotic circles around the knee. In some of the lower mammals, there is a saphenous artery. Man, being a "higher animal," depends for the maintenance and nourishment of this area on the small and variable medial malleolar branch of anterior and posterior tibial arteries. In these days of toil and stress, the main arteries themselves are sometimes hard pressed to present a palpable pulsation to the examining finger, so that their smaller branch lines fare but thinly.

The ultimate fate of the skin above the medial malleolus is obvious. Already on short rations, unsupported by the popular present-day low shoes, exposed to cold and injury, offered only stagnant venous blood by the very system which should rid it of waste products, it finally breaks down. A small injury results in an ulcer, which becomes infected, spreads, is stubborn to heal—a varicose ulcer directly related to the presence of varicose veins.

Treatment of Blood Stasis.

Treatment of this type of venous stasis in the superficial veins of the leg falls logically into three groups.

1. *Treatment for Prevention.*—Treatment is first directed toward prevention of blood stasis. Inheritance cannot be helped. However good or bad the physical equipment our forebearers have bequeathed us, it is all that we are likely to have, so we may be well advised to care for it as sensibly as possible. A certain definite amount of planned exercise should be taken by everyone every day. It goes without saying that improvement of tone and circulation in extremities is only a small part of the benefit that will accrue to such a program.

Conversely, fatigue, the deadly sodden from standing about, should be avoided. "Never stand when you can sit, never sit when you can lie down."

This often quoted rule is said to have been part of the training of the Roman soldiers. If not too soft for the Praetorian Guard, it is surely not too soft for us. It must never, of course, be used as an excuse for inertia or sheer "bone laziness."

Following any illness or operation, legs might well be used in bed much more than we now appreciate. Doctors, capable of observing, have reported that following appendectomy on themselves, they suffered more from their legs and back than from the abdomen. The "daily dozen" in bed might also avoid stagnation in the left iliac vein under the sigmoid, with the thrombosis and consequent pulmonary embolism, which shatters an otherwise happy convalescence. Children refuse to lie still on their backs in bed following operation, and in children thrombosis and pulmonary emboli are relatively rare.

Tight circular garters need not and should not be worn. Even though a small factor, they are just as well ruled out.

Pregnancy will, of course, occur, but the suggestion that legs be included in the general pre-natal care has already come from the obstetricians.

2. *Treatment to Cure.*—If blood stasis has occurred, it should and can be relieved. The dilated tortuous channels carrying a slow current of useless blood in the wrong direction, and thereby bringing about the downfall of distal tissues, should be done away with. They can be removed by surgical operation, and in this country, such was the method of choice as recently as the war years.

The injection method has now replaced operation. Almost 100 years ago, French and German workers observed independently that drugs injected into a vein sometimes caused thrombosis. This led them, deliberately, to inject irritant chemicals into varicose veins. Lord Lister is said to have injected carbolic acid into dilated veins. The results were good, but were uncertain and sometimes unpleasantly effective. Large thromboses, sloughs, and ulcers occurred. The treatment has been revived and has reached its popular position, for two reasons: first, because of the introduction of better solutions, and, second, because the principle underlying the treatment has been clearly realized and more effectively attained.

The principle, upon which depends the successful obliteration of varicose veins by injection, is not the thrombosis of the blood in the vein. In fact, the solutions used will not cause blood to clot. The blood, allowed to flow back into the syringe to prove the position of the eye of the needle within the vein lumen, does not clot. The purpose of the treatment is to produce a severe, sterile, chemical, inflammation in the intimal coat of an empty vein, so that the walls will fuse together and result in the obliteration of the vein and its gradual replacement by a fibrous cord, so small and so soft as to defy detection beneath the skin.

The technique by which this end is accomplished is simple and easy to master, but is exacting. Any errors bring prompt and unhappy consequences almost equally distressing to surgeon and patient. The technique cannot be acquired by reading a paper any more than one can learn to ride a bicycle by a correspondence course, but instruction must be included in the curriculum.

3. *Treatment to Keep Cured.*—Having obliterated the offending veins, it is desirable that their return be prevented. The inclusion of this subdivision provides opportunity to emphasize that such is not always possible. Every effort should, however, be directed toward this end. The best safeguard against recurrence of varicose veins is the complete and careful eradication of all existing varicosities and their tributaries. This does away with smaller, useless veins in which valves are incompetent and flow is reversed. If allowed to remain, these, obviously, would stretch, become large, typical, tortuous varicose veins.

A useful addition to the injection of varicosities is the ligation of the saphenous vein as it dips into the

saphenous opening to join the femoral vein. This step is often carried out, under local anaesthesia, early in the treatment of a patient who has large veins running up to the groin. It slows the downward flow in the varicosed vein and diminishes the total amount of blood in the system. Thus the chemical injected is allowed to remain longer and without so much dilution, in contact with the intima, and the effectiveness of the injections is thereby increased.

Furthermore, the division of the saphenous vein between ligatures at its extreme upper end prevents the overflow of blood from the femoral vein and so breaks the vicious circle at a most strategic point. Blood that finds its way into the deep veins is, from there on, more likely to be carried up to the heart and lungs, where its usefulness is renewed.

Treatment of Varicose Ulcers.

Most varicose veins do not cause ulcers and most ulcers of the leg are not caused by varicose veins. Ulcers are more commonly caused by injury, infection, lymph stasis, to say nothing of arterial disease, chronic inflammation, and metabolic diseases.

As has been pointed out above, however, there are some chronic ulcers of the leg which are correctly called varicose ulcers, in as much as they are produced in consequence of the disturbance in circulation which characterizes varicose veins.

These ulcers are rather small, perhaps about three centimetres across, are produced by the melting out or slow sloughing of an area of skin. The melting may be precipitated by a slight injury. Varicose ulcers are closely associated with long and often large varicose veins, which extend right up to the edge of the ulcer and even under it. Surrounding skin and leg tissues are soft, pliable, and fairly normal in colour.

Treatment is well carried out by putting such patients to bed and elevating the leg while applying to the ulcer mild antiseptic dressing, such as boracic solution or saline. By elevation, the reverse flow in the varicose veins is corrected and the tissue is no longer perfused with blood low in oxygen and glucose and high in carbon dioxide and waste products of metabolism. Under such treatment, ulcers due to varicosed veins heal steadily, even if slowly. Many patients, however, do not care to go to bed when they feel otherwise well. Indeed, many cannot afford to go to bed. Some form of ambulatory treatment is, therefore, demanded.

Ulcers of this sort can be cured, perhaps more slowly, without taking patients off their feet. Obviously, the first step in the treatment of the ulcer is to get rid of the offending veins. One proceeds, therefore, without delay to inject the varicose veins and to obliterate them from end to end. An open ulcer, which must, naturally, be infected, is no contra-indication to injection of veins. Acute infection or inflammation in the ulcer and in the surrounding skin and cellular tissue is, however, a definite contra-indication.

While injection of veins is going on week by week, the surface of the ulcer is cleansed and rendered as aseptic as possible by the use of mild antiseptic dressings, which also tend to encourage hyperaemia, if hot.

Most important of all, however, in the ambulatory treatment of ulcer is the provision of a support to the large veins of the leg and to the flabby tissues themselves.

As the deep fascia so well supports the deep system of veins, we build an artificial "deep fascia" around the superficial system. I refer, of course, to a Unna's paste bandage. These are built up layer by layer with unyielding gauze bandage and extend all the way from the base of toes to the tuberosity of the tibia, leaving out the heel for ease of application, but covering carefully well below each malleolus. The gauze is rolled on very smoothly and snugly. Rather than making reverses or darts, we cut off the bandage and lay it on again. The first coat of hot sticky Unna's paste holds it from slipping.

Such Unna's paste stockings are put on first thing in the morning, before any swelling of leg or veins has occurred, and are left on for five weeks at a time, when they are replaced, if necessary.

Obviously, varicose veins cannot be injected if they lie under Unna's paste. Therefore, we carry on the first two or three weeks with dressings to ulcer and inject the veins nearby. By the time these veins are hard, the ulcer is clean enough to cover. Injections are continued while the paste is on, either above the knee or in the other leg.

Lymph Stasis.

Stasis of blood in veins is common. Stasis of lymph in tissues is not only a common problem, but a much more serious one from the point of view of the patient. It is also a more difficult and challenging problem from the point of view of the surgeon.

Its consequences are more distressing and more varied, as well as more disabling. Other principles are involved in its treatment and in the treatment of its complications.

Lymph stasis will be discussed in a companion paper, to appear shortly.

MAJOR ARTERIAL EMBOLISM OF THE EXTREMITIES AND ITS TREATMENT

By W. L. M. KING, '37.

In this paper an attempt is being made to discuss and contrast the medical and surgical treatment of sudden embolic major arterial occlusion in the extremities—an emergency which requires immediate relief if the patient's limb is to be saved—and to set forth the chief points in its diagnosis.

Aetiology.

By definition, embolism is the carrying into the circulation of some foreign substance, such as a broken-off piece of thrombus, fat, air-bubble, septic focus, etc., the mass so carried being called an embolus.

The only type of embolus which would be large enough to produce major arterial occlusion is a dislodged thrombus or fragment of the same. Such an embolus is nearly always the result of cardiac or arterial disease leading to thrombus formation within the left heart or arterial tree. In very rare cases—paradoxical embolism—the embolus originates on the venous side of the greater circulation, but reaches the arterial side through a patent foramen ovale.

In regard to arterial disease, atheromatous placques may become detached from the wall of the aorta and lodge in a large artery lower down, but this is rare. The common cause of peripheral arterial embolism is cardiac thrombosis and the most important factor predisposing to the formation of a cardiac thrombus is auricular fibrillation. Murray, in a recent study of 30 cases of peripheral embolism, found that heart disease was responsible for the embolus formation in 83 per cent.

The embolus may break off or be dislodged in any stage of cardiac disease, but it occurs most frequently during an acute exacerbation of a chronic stage of the disease.

Sudden change in the rate of flow of blood, such as occurs in a fibrillating patient when his heart suddenly becomes irregular through the use of digitalis or quindine, seems to be the agent which precipitates this dislodgement in very many cases.

Let us pause to consider very briefly the main factors in thrombus formation and how they may operate within the chambers of the heart. They are three:

1. *Primary injury to endothelium.*—It is essential that there be some break in the smooth endocardial membrane lining the chamber before a thrombus will form. Such a break acts as a nidus for

the platelets to settle about. Bacterial destruction is nearly always the cause of this injury. Here lies one of the strong connecting links between peripheral arterial embolism and organic disease of the left heart. The organism causing the rheumatic lesions in the heart produces the necessary injury to the chamber wall and so paves the way for a thrombus to form. In the same way, bacterial infection in the wall of the aorta or a large artery may involve the endothelium and damage it.

2. *Change in rate of blood flow through the heart.*—Slowing of or obstruction to the blood flow, especially when it results in the formation of eddies, allows the platelets to settle out and agglutinate. Some believe that both this factor and endocardial damage must be present before a thrombus will form. One may realize from this why auricular fibrillation is so important a factor in peripheral embolism—the platelets tend to become precipitated and agglutinated by the greatly decreased (almost static) blood flow in the fibrillating auricles or by eddies produced by leaking and deformed valves. Embolism follows when a sudden change to a normal rhythm occurs, the force of the normal auricular contraction wave serving to force this "ball" thrombus through the mitral valve into the systemic arterial circulation.

3. *Changes in physical and chemical properties of the blood.*—Such changes may be an increase or decrease in the viscosity of the blood, alteration in quantity or quality of the platelets, infection, metabolic disease, some debilitating disease or peripheral arterial disease itself. It is not likely that any of these things has much to do with intracardiac thrombus formation. The main effect of these changes is to alter the platelets quantitatively and qualitatively, so as to favour agglutination — and anything favouring agglutination will be a large factor in thrombus formation.

Another event which often gives rise to embolism is mural thrombosis occuring on the endocardium of a portion of devitalized or dead heart mucle, caused by a shutting off of the blood supply by coronary sclerosis with thrombosis. Such a thrombus is often easily dislodged by any increase, especially if sudden, of the force of the heart beat.

Pathology.

Rheumatic heart disease is the major cause of intracardiac thrombus formation. Inflammation of the endocardium is the source of injury to it. Slowing of the blood stream is the result of cardiac decompensation, due to myocardial involvement or auricular fibrillation, or to deformity of mitral and aortic valves, which causes obstruction to the flow of blood and gives rise to eddies.

This stasis of the circulation allows the platelets to settle out and become agglutinated and attached to any minute injury of the endocardium. As the deposit of platelets grows, it entangles first red and later white cells. Eventually, endothelial cells are injured and fibrin ferment liberated, producing fibrin. A large mass soon forms which becomes quickly organized. Due to its rate of growth, its disproportion between surface attachment and bulk, and to derangement of the heart mechanism, this thrombus becomes dislodged into the systemic circulation as an embolus.

Due to the rapid decrease in size of a vessel ,when large branches are given off, this embolus tends to lodge at bifurcations, the commonest of all being those of the aorta and the common iliac arteries. Passage of the embolus into the large branches of the arch is relatively uncommon. Most emboli doing this pass along the external iliac (because it is a direct extension of the common iliac) and wedge at its bifurcation into femoral and profundus branches.

The irritation produced by the embolus results in constrictor spasm of the vessel distal to it and this may completely occlude the lumen. This is probably the origin of the initial severe pains being augmented by the pain of anoxaemia which follows. If obstruction is not complete, a little blood gets past and results in the deposition of

platelets distal to the embolus, producing secondary thrombosis, which soon completes the obstruction.

The results of occlusion depend upon the following factors:

1. Size of occluded vessel.
2. Its situation.
3. The number of plugged branches.
4. The efficiency of the heart.
5. The number of available, dilatable, collaterals.

The usual consequence is infarction of the organ supplied or gangrene of the extremity.

Signs and Symptoms.

1. *Pain.* — This is usually the first evidence of the accident and may be anywhere from an indefinite ache to an excruciating, localized, sudden pain propagated along the course of the vessel involved. The former is due to incomplete blockage and is the result of tissue anoxaemia. The latter is the result of sudden, violent arteriospasm, produced by immediate complete blocking. In partial occlusion, pain becomes more severe and constant as secondary thrombosis ensues. In cases where secondary thrombosis develops gradually or there is embolic obstruction at the bifurcation of aorta or common iliacs, the symptoms at onset are tingling, numbness and coldness. In the latter case, this has been accounted for by the fact that these large vessels have relatively little muscular tissue in their mediae, compared with the smaller peripheral arteries, but more fibro-elastic tissue which cannot go into spasm. Therefore, numbness and tingling are far graver symptoms prognostically than is agonizing pain.

2. *Paraesthesiae.*—i.e., numbness and tingling, etc. The significance of these has been pointed out already.

3. *Skin colour changes.*—Generalized cyanosis of the lower extremities is a distinguishing feature of major arterial embolism. The absence of pallor or of cyanosis and mottling means that the limb is not in danger of death, due to anoxaemia. In sudden complete block, there is usually pallor of the distal part, a cyanotic band farther up, and an area of blotchy mottling above that. This is of value in localizing the block if one takes into account the fact that normal skin color extends considerably below the point of blockage.

4. *Temperature Changes* — correspond to areas of color change, being coldest where pallor is greatest.

5. *Loss of Pulsation.*—This is a most valuable localizing sign, because it corresponds very closely with the level of obstruction. The instrument used for detecting this is the oscillometer.

Sensory changes, paralysis and general constitutional changes are other less important findings. Arteriographs for localization of the embolus by the X-ray are as yet in the early stages of development.

Diagnosis.

This depends upon the sudden pain or indefinite ache, on the skin color and temperature changes, and upon the loss of pulsation. Differentiation may have to be made from venous thrombosis, arteriosclerotic gangrene, Buerger's disease, and Raynaud's disease.

Treatment.

(a) *Medical.*—The general objects of medical therapy for major arterial occlusion are:

1. Relief of pain.
2. Re-establishment of an adequate collateral arterial circulation.
3. Prevention of infection of poorly nourished tissues.

After many years' research by various investigators, Hermann and Reid of Ohio perfected an apparatus which has been used successfully all over the world in the treatment of arterial embolism. Their machine is called "Pavaex," a term coined from Passive Vascular Exercise. A glass boot is applied to the patient's extremity and is made to fit snugly by a rubber cuff. Air is let into and exhausted from the boot at a rate of two to four times a minute, so regulated that a positive pressure of 20 mms. Hg. alternates with a negative one of 80 mms. The leg and boot are elevated a foot above the heart level to aid the venous return and air, preheated to 104° F., is

introduced to aid in dilatation of the collateral vessels by local hyperthermia.

The length of the treatment depends on the severity and urgency for restoration of the circulation. In cases of sudden arterial occlusion, treatment is continuous for 70—100 hours, with rests of only an hour or two in 24 hours, or until the circulation is restored. The usual rate for most cases is, however, 100 hours at five hours a day.

The great advantage of Pavaex is that it overcomes the temporary vasospasm and puts a quick end to the agonizing pain. Relief of the pain is, therefore, almost immediate. It also increases greatly in size and number the useful collateral arteries of distal parts of the extremity.

The use of Pavaex therapy is absolutely contra-indicated by the presence of an acute or sub-acute thrombophlebitis, Buerger's disease, or arteriosclerosis. It is of little value in cases of angiospasm of central origin or of inflammatory disease involving the small vessels.

(b) *Surgical.* — Many investigators are of the belief that the ideal treatment of major arterial embolism is immediate arteriotomy, removal of the clot and suture of the vessel. It is necessary that the operation be done as early as possible, for not only is removal easier, but there is less danger of secondary thrombois, due to injury of the vessel wall. The following factors have much influence on the prognosis following embolectomy:

1. Blood pressure.
2. Velocity of the blood.
3. Locus of the infection.
4. Completeness of obstruction.

The first step in pre-operative treatment, therefore, is maintenance of blood pressure and rate of blood flow. Local anaesthesia is preferred to general, in order that the surgeon may better gauge the result of his work. The vessel is exposed, incised, and the clot withdrawn, bleeding being controlled by rubber tubing held lightly in the fingers. The opening is closed by fine, oiled, continuous silk suture, after flushing out the wound with fresh blood. No drainage is needed. After operation, the extremity is kept warm and at rest.

Embolectomy is contra-indicated by bad general condition of the patient, by multiple embolism in the organs, by lapse of too great a time between occurrence of embolism and operation, and by uncertainty of diagnosis.

The results vary with the time between accident and operation. The time required for secondary thrombosis to occur is 12—48 hours. From recorded cases, it seems that a lapse of greater than six hours makes the outlook very unfavorable.

Whether this form of treatment is to be preferred to the Pavaex method is very difficult to say. The risk of the operation is great, yet if it succeeds, the patient has the use of his main artery again. If it does not succeed, one may still resort to Pavaex treatment. On the other hand, Pavaex therapy is far less shocking to the patient, gives immediate relief from pain and opens up many useful vascular channels in the limb. For this reason, a second embolus following the first will not produce the same damage. Where embolectomy has been performed, there is every chance of an embolus again lodging in that artery. The underlying heart condition giving rise to the embolus formation is one that offers the constant threat of formation of new emboli, so that this should be taken into consideration when embolectomy is contemplated.

Both methods of treatment are so recent that much more time is required and a more complete series of case reports needed before one can be in a position to state which method is the sounder.

APPLICATIONS in writing will be received by the Editor up until November 23, for the following positions:

ASSISTANT EDITORS

from the Vth and IVth years in Medicine and IVth year Biological and Medical Sciences Course.

PHYSIOLOGY OF CEREBRAL CIRCULATION

By FRANK B. THOMPSON, B.A., '39.

This subject may best be introduced by a brief consideration of the Munro Kellie doctrine, a theory that arose at the end of the eighteenth century. This theory stated that the skull being a rigid box, and the solid contents of the skull being constant in amount, then the volume of the fluid contents of the skull must be constant also. Therefore, although the total contents must be constant the quantity of the blood or the cerebro-spinal fluid could vary provided only that the two changed simultaneously and in inverse proportion.

Weed investigated the relation between intracranial pressure and osmotic pressure of the blood. He found that injection of a hypertonic solution caused a negative intracranial pressure, due to a dehydration of the brain. It was assumed that there must be a corresponding increase in the intracranial blood volume and therefore a dilatation of the cerebral vessels. Wolf and Forves, however, showed that the decrease in volume of the cerebro-spinal fluid, resulting from the injection of hypertonic solutions, was accompanied by a constriction of the pial vessels, and if it could be shown that the "cerebral vessels," those passing through the brain substance itself, did the same thing, then, of course, it would be proof that the Munro Kellie doctrine was wrong. By using a colour photography method whereby changes in the blood vessels of the brain itself could be seen and recorded, Kubie and Hetler showed that intravenous injection of hypertonic solutions is followed by a narrowing of the pial vessels and a dilation of the "cerebral vessels" or vessels of the cortex, and that the reverse occurred when hypotonic solutions were injected.

Those experiments clearly demonstrated that the decrease in volume of cerebro-spinal fluid, following the injection of hypertonic solutions into the blood stream, is accompanied by an increase in the intracranial blood flow; and therefore support the Munro Kellie doctrine which the work on the changes in the pial vessels had indicated might be wrong.

One might ask why there should be dilatation in one case and constriction in the other? Kubie and Hetler suggest that the pia has arterioles and venules and few if any capillaries, and that the constriction here is due to a direct action, by the change in salt concentration of the blood, and also to a lessened peripheral resistence in the cortex; whereas the cortex has a lot of capillaries and the dilatation is probably a direct response to diminished fluid contents within the head, and due to the negative intracranial pressure.

Now to consider the influences on cerebral circulation of variations in the chemical properties of the blood, and especially in the amounts of carbon dioxide and oxygen present.

The first important work on this showed that a decreased volume of blood caused an increased alkalinity of arterial blood and an increased acidity of venous blood, and that these changes were independent of respiratory changes. This indicated that either volume flow or ventilation changes can alter the pH of the blood in the "cerebral vessels."

Bronk and Gesell, in a 1927 paper state, that "granting a co-ordinated function of the respiratory and circulatory systems, and a dependence of functional behaviour of the respiratory centre on variations in its metabolism, it seems as logical to propose a control of circulation dependent on variations in the metabolism of the circulatory centres. For just as surely as pulmonary ventilation influences the acidity on both sides of the neurone membrane inside and outside the

cell, so circulation influences these same chemical conditions." Then on the assumption that these acid relations were in turn significant in circulatory as well as respiratory control, they investigated the effects of changes in pH of the cerebral arterial blood on the volume of the cerebral blood flow. Using a continuous thermo-electric method they concluded that:

1. The administration of carbon dioxide in the respired air causes an increased carotid flow and a decreased femoral flow.

2. That variations in the magnitude of artificial respiration has the same effect.

3. That the changes in carotid flow were not dependent on changes in the mean blood pressure. (Since this was kept constant).

4. That the reciprocal relation between carotid and femoral flow, suggests that circulatory adjustments following an increase in the carbon dioxide content of the blood occur in favour of the brain.

Bernthel and Gesell studied the effect of sodium cyanide on cerebral blood flow and found that it had an effect similar to that of carbon dioxide. Other work by Schmidt showed that the "cerebral vessels" are under intrinsic chemical control, for they are dilated by carbon dioxide, anoxaemia, acid, heat and cerebral anemia; and constricted by excess oxygen, alkali, cold and probably by an increased blood flow. These results indicate the intimate functional relationship that exists between chemical composition of the blood and the volume of the cerebral blood flow.

Wolf and Lennox made a study of the effect on the pial vessels, of variations in the oxygen content of the blood. Their work proved that during anoxaemia there is an increase in the cerebral blood flow. Lennox and Gibbs using human subjects confirmed all the facts shown to be true in animals and concluded that the circulatory changes in the brain and leg in response to gas changes in the blood were opposite, and that in man, as well as in animals, the brain benefited by this "preferential treatment." Their experiments showed that in general the circulatory responses to chemical stimuli are greater in the brain than leg, one reason being that the arterial venous difference is greater for the brain than for the extremities, and therefore there is a greater opportunity for an improvement in the quality of the blood passing through the brain to occur. Also that cerebral circulatory responses are more constant than those of the leg, considered from time to time and from subject to subject.

Dr. Irving and Mrs. Welch investigated the nature of the "preferential control" of circulation in the dog, and concluded that during carbon dioxide inhalation, the respiratory quotient of the brain is much larger than that in the leg. The respiratory quotent indicates the process of equilibration of the tissue with a change in pressure of carbon dioxide, and shows that the pressure of carbon dioxide changes more quickly in the brain than in the leg. They believe that the greater respiratory quotient of the brain indicates that the pressure of carbon dioxide was greater here than in the muscles, and that the brain was giving off carbon dioxide to the blood whereas the legs were still taking it up, and that, if the stimulus to differential control is the pressure of carbon dioxide, then this is exerted more in the brain. However, they point out that the carbon dioxide pressure increases in both leg and brain and yet the flow decreases in the leg. Therefore, they conclude that the differential control cannot be by a reflex process or a system intrinsic in the vascular bed. Their final conclusion is, that the stimulus for differential control may originate in the brain itself, but the results may be reinforced by, or it may even originate in, a site extrinsic to the brain.

It remains to consider what part the nervous system plays in the control of the "cerebral vessels." Early work led to the belief that there were no vasomotor nerves to the cerebral vessels and that the cerebral circulation pas-

sively followed changes in blood pressure. However, in 1928, Forbes and Wolf showed that cervical sympathetic stimulation caused constriction and vagal stimulation dilatation and so the existence of a vasomotor control over the "cerebral vessels" was established.

Further work has indicated that the pathway of the sympathetic fibres is from the superior cervical ganglion along the carotid and from the inferior cervical ganglion along the vertebral. The pathway of the vagal dilatory fibres is up this nerve to both facial nuclei in the medulla, out by facial nerves towards the geniculate ganglion just proximal to which the fibres separated to join the nervus intermedius of Wrisburg. From this nerve they pass into the greater superficial petrosal nerve to reach the plexus surrounding the carotid arteries. These nerves convey active bi-lateral, dilator impulses.

It was found that there, as was the case in considering the effect of chemical changes in the blood, when the dural vessels dilated the pial vessels constricted. This is a compensatory mechanism to maintain a constant intracranial prussure.

Vasomotor control to the medulla, hypothalamus and cortex is very weak and chemical regulation of the calibre of the vessels in these and probably in all the cerebral vessels is much more important than vasomotor control. The constriction due to sympathetic stimulation is very slow in onset, it develops slowly and recovery is slow, all of which would suggest a humoral mechanism.

The action of various chemical substances and extracts on the cerebral vessels has been determined. It is found that epinephrine and pitressin cause a sharp rise in arterial and intracranial pressures, and vasoconstriction. Amyl nitrite, pituitary extracts and histamine cause vasodilatation with increased cerebro-spinal fluid pressure. The vessels are very sensitive to histamine, causing them to dilate.

In all experiments on the influence of chemical changes in the blood, vasomotor control, or the effect of drugs on the cerebral circulation, the level of the systemic blood pressure must be kept constant if the results are to mean anything. Changes in the level of the systemic blood pressure cause a marked change in the cerebral blood flow which may or may not be accompanied by a change in tone of the cerebral vessels.

Work done in recent years brings out the fact that the physiology of the cerebral circulation is not markedly different from that of the circulation in the rest of the body, but has some characteristic features. It is evident that under conditions of stress, where there is a greater carbon dioxide content or a decreased oxygen content of the blood, there are changes that take place in the distribution of the blood, such as favour the brain. The mechanism whereby this reciprocal relation in carotid and femoral flow is established, has remained obscure up to the present time. There is little doubt but that the central nervous system is the controlling factor and it seems probable that the seat of control lies in the medulla. Also it would seem that the amount of blood flowing in the cerebral vessels at any time depends upon four separate physiological factors.

1. Systemic blood pressure.
2. Chemical constitution of the blood with respect to carbon dioxide, oxygen, cyanide, adrenalin, drugs.
3. Osmotic pressure of the blood.
4. Vasomotor control (sympathetic and para-sympathetic).

The relative importance of these factors is a debatable question, but it is quite certain that the first is by far the most important. Of the other three, their functional significance is doubtful, but it would appear that the vasomotor control is probably the least important. Certainly it has less functional significance here than elsewhere in the body. There is not the need of such an adequate control because the flow of blood to the head is carefully governed by reflexes arising in the aortic arch and carotid sinus areas and protecting the brain from changes in blood pressure, carbon dioxide, oxygen, and hydrogen ion

concentration in the blood. It would seem, therefore, that although the physiology of the cerebral circulation is similar to that of the circulation in the rest of the body, its real control is found in the physiological mechanisms controlling the systemic circulation and acting indirectly, therefore, on the cerebral circulation.

Reference

R. C. Smith—"Nervous Control of Cerebral Circulation"—A paper to the Physiological Journal Club.

EMBRYOLOGY OF THE BLOOD VASCULAR SYSTEM

By JAMES G. WATT, '40.

Blood Islands.

The precursors of the blood corpuscles and blood vessels first make their appearance in the third week of fetal life. They appear from the mesoderm of the wall of the yolk sac, in the body stalk, and in the chorion. Certain cells arrange themselves into clusters or cords. These are called angioblasts. The angioblasts on the periphery of the cluster give rise to the endothelium, which is a layer of flattened cells. Those in the centre of the cluster form a clear, secreted fluid, the plasma, and the earliest blood corpuscles. These isolated areas are termed blood islands. By growth and union, these vascular areas are converted into a plexus of blood vessels on the yolk sac, called the "area vasculosa."

Vessels appear in the embryo itself just before the earliest somites. They arise from the mesoderm of the embryo, not from the extra-embryonic mesoderm, yet many are continuous with the vessels on the yolk sac. New vessels now arise as outgrowths of the primitive vessels previously formed. These join with neighbouring vessels to form a close network.

Early Vessels.

The veins and arteries are not structurally distinguishable at first, since they do not receive their accessory coats until the twelfth week of fetal life. The neighbouring mesenchyme lays down these coats around the endothelium, which is the primary tissue in the walls of the vessels.

The definite vessels arise through selection, enlargement, and differentiation of appropriate paths in the network of primary capillaries. The capillaries from which the flow has been diverted atrophy. This selection depends to a great extent upon heredity. The fundamental plan is a paired symmetry, but this bilateral arrangement is obscured later by the fusion of some vessels and the atrophy of others.

In an embryo of four weeks, paired unfused heart tubes and paired ventral and dorsal aortae appear. The ventral and dorsal aortae connect through a primitive first aortic arch. Umbilical arteries arise from the dorsal aortae and pass into the body stalk to ramify in the chorion. The chorionic blood is returned to the embryo through umbilical veins, which course in the body wall.

Soon the heart tubes fuse to form a single tube. Vitelline arteries arise, which interconnect the dorsal aortae and the yolk sac. Vitelline veins return blood from here to the primitive heart, and open into it at the caudal end.

At this stage, the heart is anterior to the foregut, and is situated in the pharangeal region. From its cephalic end, two aortae arise, which pass through the mandibular arches as the first aortic arches to become the dorsal aortae. Caudal to the first aortic arches, five additional pairs develop, in association with the remaining five

visceral arches. Four veins enter into the caudal end of the heart, two vitelline veins, and two umbilical veins.

The next important step is the formation within the fetal body proper of paired veins. They are paired precardinal veins, which drain blood from the head region, and paired postcardinal veins, which serve the posterior part of the body. Before entering the caudal part of the heart, now the sinus venosus, the two veins of each side unite into a common cardinal vein (Duct of Cuvier).

Soon the two dorsal aortae fuse into a single descending aorta. Three important ventral, vitelline arteries now arise, which are destined to become:

(1) The coeliac to the stomach and pancreas region.
(2) The superior mesenteric to the region of the small intestine.
(3) The inferior mesenteric to the region of the large intestine.

Development of the Exterior of the Heart.

In the pericardial region are the two primitive primordia of the heart. They are thin-walled endothelial lined tubes flanked by splanchnic mesoderm and separated from each other by the entoderm and the cavity which it encloses. As the fore-gut closes, it allows the two tubes to swing together and fuse. The wall between the two breaks down so that a single tube is formed. The endothelium forms the endocardium, and the mesoderm the myocardium and epicardium of the walls of the heart. At this stage, then, the heart is a double-walled tube suspended by a temporary dorsal mesentery, termed the dorsal mesocardium. In mammals, including man, there is no ventral mesocardium.

Constrictions appear in this simple tubular heart before fusion is complete. They divide it from cephalad to caudal into bulbus arteriosus, ventricle and atrium respectively. A fourth division arises by constriction of the hind end of the atrium. It is the sinus venosus.

The sinus venosus receives all the venous blood from the pre-cardinal and post-cardinal, umbilical and vitelline veins. The blood is then transmitted from here into the atrium, where it is pumped into the ventricle. This latter chamber forces it through the bulbus into the ventral aortae.

The heart then twists into an "S" shape. The bulbus and ventricle drop ventrally and caudally. The atrium and sinus venosus assume a more dorsal and cephalic position. Thus, the original position of the chambers of the heart is now reversed.

The atrium being constricted dorsally by the gut and ventrally by the bulbus, can only expand laterally. Two sacculations form, which become the left and right atria. Superficially, the separation is marked by the interatrial sulcus. Due to the growth of the liver, the flow of venous blood from the trunk is shifted to the right side. This causes the right side of the sinus venosus to enlarge more rapidly than the left.

The primitive bulbus becomes incorporated into the ventricle, and this chamber becomes separated from the atria by a deep coronary sulcus. A median longtitudinal groove appears on the ventricle. This is the interventricular sulcus, and marks the position of the internal septum already partitioning the ventricle.

Development of the Interior of the Heart.

The important changes which lead to the formation of a four-chambered heart are:

(1) The partitioning of the common atrium into right or left chambers.
(2) The division of the atrio-ventricular canal into two canals.
(3) The incorporation of the sinus venosus into the right atrium.
(4) The division of the ventricle into two chambers, a right and a left.
(5) The division of the bulbus into the aortae and pulmonary arteries.
(6) The development of valves.

Two endocardial thickenings occur on the walls of the common atrio-ven-

tricular canal. They are termed endocardinal cushions. These fuse and divide the atrio-ventricular canal into two canals, a right and a left.

At the same time, a thin membrane begins to grow from the mid-dorsal wall of the atrium. It is named the septum primum, and it grows toward the atrio-ventricular canals, to fuse with the endocardial cushions. This divides the original chamber into two separate ones, but the septum primum becomes thin and perforates, thus establishing communication between the two atria. This opening is called the interatrial foramen.

A second septum arises just to the right of the septum primum. This contains a prominent opening, named the foramen ovale. The two septa grow in such a way that the free edge of the septum primum overlaps the foramen ovale in the septum secundum. It serves as a flap-like valve, which permits blood to pass from the right to the left atrium, but not in the reverse direction. This condition exists until after birth.

The two largest veins in the body, the superior and inferior venae cavae, have now been formed. They both drain into the right horn of the sinus venosus. The atria increase rapidly in size and the sinus becomes merged into the right atrium. This action causes the superior venae cavae to drain directly into the upper part of the atrium and the inferior venae cavae directly into the lower part. The left horn and transverse part of the sinus receives the veins of the heart itself and becomes the coronary sinus, which opens into the posterior wall of the right atrium.

Two valvular folds existed in the sinus venosus, where it opened into the dorsal wall of the right atrium. Part of the left valve becomes continuous with the septum primum, and it may give rise to the septum secundum. The right valve becomes a rudimentary crest, the crista terminalis, and also the valve of the inferior venae cavae and the valve of the coronary sinus.

Originally, a single pulmonary vein drains into the left atrium. This vessel bifurcates into right and left veins, which in turn divide into two. As the atrium grows, these vessels are drawn into the wall, so that two—then eventually four—pulmonary veins open into the left atrium.

Two longtitudinal thickenings arise in the bulbus. They meet and fuse, thus dividing the bulbus into aortic and pulmonary trunks. The septum follows a spiral course, so that the two vessels are wound about each other. The pulmonary trunk crosses ventral to the aorta. Proximally the spiral division forms in such a way that the pulmonary trunk opens into the right ventricle and the aorta into the left. Semilunar valves form in both the aorta and pulmonary arteries at their junctions with the ventricles.

Coincident with the division of the bulbus, the interventricular septum arises on the base of the single ventricular cavity. It extends to the ventral endocardinal cushion. For a time, it is incomplete and there is a communication between the right and left sides through an interventricular foramen. Later this septum and the endocardial cushions fuse to close off the foramen.

Elevated folds of endocardium arise from the margins of the atrio-ventricular canals. They become invaded by muscle and attached to trabeculae of the wall. These cusps are formed about the right atrio-ventricular orifice. These become the tricuspid valve. Two cusps only form about the left orifice, to give rise to the mitral valve. The cusps become tough and fibrous and connect with chordae tendineal, similarly transformed from muscular cords. These latter continue into the papillary muscles.

Development of the Arteries.

As mentioned previously, six pairs of aortic arches develop, but all are not present at the same time. This is due to the fact that some are going through degenerative changes, while others are just coming into existence. The first arch appears first, the sixth last.

The first and second pairs of aortic arches drop out early and are replaced

by vessels which do not communicate with the ventral aorta. The dorsal aorta, however, persist at this level, but between the third and fourth arches, both vessels atrophy. A continuous vessel beginning with the third arch and continued into the head region by that remaining part of the dorsal aorta results. These vessels are the external carotid arteries. The external carotids arise from the ventral aorta, but transfer themselves on to the third arches. Thus part of each third arch becomes a common carotid artery.

The left fourth arch and part of the ventral aorta, now called aortic sac, become the permanent aortic arch. This arch enlarges greatly. The right fourth arch elongates into the innominate artery. In so doing, it becomes the main stem for both the subclavian and common carotid artery of the right side.

The fifth arches are inconstant, often incomplete and transitory. They disappear without leaving a trace.

The sixth arches come into being by a sprout from each dorsal aorta bridging across to the primitive pulmonary arteries. The connection on the right side disappears again, but that on the left persists as the ductus arteriosus until after birth.

The bulbus is dividing at this time and it does so in such a way that the aortic trunk opens into the third and fourth arches, whereas the pulmonary trunk is directed into the sixth arches.

The dorsal aorta terminates caudally as the middle sacral artery. Throughout its length are dorsal, lateral and ventral branches in three rows. The dorsal branches of the first six cervical segments fuse to form the vertebral artery. In the thoracic region, the ventral rami of the dorsal branches give rise to the intercostal arteries, and in the lumbar region to the lumbar arteries. The lateral branches are not segmentally arranged. They supply the sex glands, suprarenal glands, and the metanephric kidneys. The ventral branches are reduced until they appear only as the coeliac and the superior and inferior mesenteric arteries.

A bud extends from the subclavian into the upper arm, which becomes the brachial artery.

A branch, called the sciatic, is given off from the umbilical (future hypogastric), and for a while this is the chief arterial stem of the lower limb. A new stem buds off the umbilical, which is the common iliac. It divides into internal and external iliacs. A branch of the latter, the femoral, supercedes the sciatic. The sciatic persists proximally as the inferior gluteal artery only.

Development of the Veins.

There are three systems of paired veins in the embryo now: (1) The umbilical veins from the chorion; (2) the vitelline veins from the yolk sac; (3) the post-cardinal and pre-cardinal veins in the wall of the embryo itself. The pre-cardinal and post-cardinal vein of each side unite into a common channel, called the duct of Cuvier. This duct opens into the horn of the sinus venosus. At this stage, then, three veins open into the right horn of the sinus and three into the left.

The liver grows in the path of the vitelline vessels. About the fifth week, these vessels resolve into a plexus of veins within the liver. The proximal part of the left vitelline vein becomes absorbed in the sinusoids of the liver and this causes a shift in its flow of blood to the right horn of the sinus venosus. This new channel becomes the left hapatic vein. The corresponding part of the right vitelline becomes the right hepatic. Caudal to the liver, the vitelline veins form a figure of eight about the gut. In the upper loop, the left part degenerates, and in the lower loop, the right part atrophies. In this way, the portal vein is formed.

The liver tissue continues to expand and comes into contact with the umbilical veins, which run in the body wall. Their blood becomes diverted so that it flows through the sinusoids of the liver to the heart. Blood from the left one flows diagonally to the right horn of the sinus venosus. Soon the entire right umbilical, and the part of the left proximal to the liver, atrophy.

The left is fairly large and runs in the free edge of the falciform ligament.

A new and more direct channel forms from the liver to the sinus. This is the ductus venosus. It is directly in line with the left umbilical vein. Both the left umbilical vein and the ductus venosus atrophy after birth.

Each precardinal vein consists of a primary head vein and the true precardinal. Primary head veins have three pairs of tributaries, which extend over the head, as the various sinuses of the brain.

The true precardinals run caudad as the internal jugular veins. A communicating channel arises between the left and right sides. It becomes the left innominate vein. The remainder of the left precardinal below the innominate persists as the highest intercostal vein, and the inconstant oblique vein of the heart. The right precardinal and common cardinal become the superior vena cava, below the point where the left innominate enters. Above this anastomosis and extending as high as the right subclavian vein, the right precardinal is named the right innominate vein.

Three sets of veins are developed caudal to the heart for the purpose of draining legs, body wall, and some of the viscera.

The first set, the post-cardinals appear as this organ wanes. They run dorsal to the mesonephroi and receive tributaries from the legs and body wall. An early anastomosis occurs between them where the veins from the legs enter into them. This connection persists as a part of the longer and more oblique left common iliac vein. On the right side, part of the right post-cardinal persists as the arch of the azygos vein.

The sub-cardinals are the second set. They lie ventro-median to the mesonephroi and connect through the sinusoids of this organ with the post-cardinals. They also anastomose with each other. One of the connections between them becomes the stem, uniting the left renal vein to the vena cava. They also give rise to the paired suprarenal and sex veins. The right sub-cardinal forms a segment of the inferior vena cava.

The third set are the supra-cardinals. They replace the post-cardinals, but become broken in the region of the kidneys. They give rise to the azygos and hemiazygos veins. The right supra-cardinal forms the caudal most part of the inferior vena cava.

The unpaired inferior vena cava then is formed from several sources.

(1) An hepatic segment from the hepatic sinusoids and the hepatic vein.
(2) A prerenal segment from the right subcardinal.
(3) A renal segment comprising an anastomosis between the right sub-cardinal and right supra-cardinal veins.
(4) A supra-cardinal portion from the right supra-cardinal in the lumbar region.

Fetal Circulation and Changes at Birth.

During fetal life, oxygenated blood returning from the placenta enters the embryo through the umbilical vein. It flows through the ductus venosus and liver sinusoids and hepatic veins into the inferior vena cava. It is mixed here with the impure blood of the inferior vena cava and portal vein. This contaminates the pure placental blood only slightly and it enters the right atrium relatively unchanged. The impure blood of the superior vena cava also enters the right atrium.

Most of this mixture passes through the foramen ovale into the left atrium. Very little as yet goes into the right ventricle, since the lungs are not in use in the fetus. The blood then enters the left ventricle, and passes out through the aorta to the head trunk and limbs. A large part of it passes back to the placenta through the umbilical arteries.

The blood that does enter the right ventricle is passed out through the pulmonary arteries to keep the lungs alive. However, a large part of this is short-circuited through the ductus arteriosus into the aorta without reaching the lungs.

At birth, the placental circulation ceases quickly and the lungs become functional. This changes greatly the character of blood in the large vessels and the umbilical vessels are thrown into disuse.

The lungs expand, but do not immediately take over full functional activity. The equalizing of the blood intake of the two atria must await the gradual enlargement of the pulmonary vessels, in response to their new functional activity. Thus the flow of blood through the foramen ovale is not abandoned abruptly after birth. It requires about two months for the complete functional closure of the foramen ovale. The two septa do not fuse anatomically for close onto a year. It is interesting to note that 25 per cent. of all individuals never obtain complete closure. The sight of the foramen is permanently marked by the fossa ovalis.

The flow of blood through the ductus arteriosus discontinues, and in about three months it becomes impervious. It persists as a solid cord the ligamentum arteriosum.

The umbilical vessels are thrown into complete disuse after birth. The vein becomes fibrous tissue and persists as the cord-like ligamentum teres of the liver. Sometimes a small vein remains. The two arteries become the lateral umbilical ligaments. The ductus venosus also atrophies, but remains marked by the ligamentum venosum, which is superficially imbedded in the wall of the liver.

The True Economy of Dextri-Maltose.

It is interesting to note that a fair average of the length of time an infant receives Dextri-Maltose is five months: That these five months are the most critical of the baby's life: That the difference in cost to the mother between Dextri-Maltose and the very cheapest carbohydrate, at most is only $6 for this entire period—a few cents a day: That, in the end, it costs the mother less to employ regular medical attendance for the baby than to attempt to do her own feeding, which in numerous cases leads to a seriously sick baby eventually requiring the most costly medical attendance.

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STATE HEALTH INSURANCE

By W. K. KERR, '40.

Introduction.

The last hundred years in the history of medicine have witnessed, not only a tremendous advance in the knowledge of medical subjects, but also a notable change in the general subject studied.

While always concerned with the human body, the medical profession has progressively turned its attention from the cure of diseases of the human body to the prevention of such diseases, and more recently the emphasis is being shifted from the negative aspect of diseases to the more positive aspect of promoting health.

Approximately coincident and parallel with these developments in medicine is the advent of our industrialized society, with its higher standards of living for all, its economic insecurity for a multitude of wage-earners and with unemployment and poverty for one-tenth of our population.

These changes in medicine and in economics are in part responsible for the financial relations between physician and patient to-day, financial relations of such an insecure and unsatisfactory nature that a re-organization of those relations has become imperative.

Before considering health insurance, it would be well to examine briefly the present organization of medical care.

From the point of view of medical services, the population may be divided into the following groups:

1. The Indigent, who are unable to pay, and for whom some provision is usually made.

2. The Well-to-do, who can secure such medical care as they require, because they are able to pay for it.

3. The so-called Middle-class, whose incomes vary greatly, but who, on the whole, are presumably able to provide for themselves with the necessities of life, including medical care.

The Medical Care of Indigents.—In general, the care of indigents in their homes is left to the medical profession, although, in some places, a physician is paid out of public funds to provide such care. When hospital care is required, a per caput per diem allowance is usually paid to the instiution caring for the patient, but no fee is paid to the physician.

The members of the profession, in general, are willing to give a certain amount of free service. Nevertheless, there is a general feeling among them that the profession is being exploited and that the governments are meeting their responsibilities for the care of the indigent at the expense of the medical profession.

It is also true that, under the present system, the indigent must perforce accept what is offered. This state of affairs is not satisfactory, either to the profession or to the indigent patient.

Medical Care of the Well-to-do.—This group presents a problem because there is no fixed charge for the services purchased. The charge mounts according to the ability to pay and simply because these individuals have more money, they are expected to pay more than anyone else, and so compensate in some measure for those who pay less than the cost.

This is not a sound economic scheme. There is no reason why anyone should be overcharged, simply because he can afford to pay for such overcharge.

Medical Care of the Middle-class.—The income of this group varies within a wide range. An attempt has been made to meet the needs of this group by a sliding scale of fees, adjusted to the presumed ability of the individual to pay.

The method naturally tends to penalize the thrifty, who, because of their thrift, appear to be and actually are more capable of paying. Those who pay assume at least part of the burden of those who will not or who are unwilling to pay, and who, in one way or another, avoid paying their bills.

Criticism.

The proportion of well-to-do, middle-class and indigent patients is not the same for all practitioners, so that the doctor who does most free work, may have little or no share of the well-to-do patients. A condition then prevails, under which the medical care of the indigent falls, not only on the medical profession as a whole, but often on that part of the profession which can least afford it.

It is obvious that the chief objection to the present system of medical practice is the financial problem it presents, both to patient and physician. The average cost of medical care per year per adult is $20-$40. But the total cost for the whole population is unevenly distributed, so that it falls on a few individuals at unpredictable times.

The amount of illness does not vary with family income, but the cost of medical care does.

Modern medicine is becoming increasingly more costly with the development of hospitalization and specialist services, and these facilities fail to reach sparsely populated areas.

All of these factors contribute to a condition under which many are going without medical care, some are overwhelmed with the costs and losses due to illness and preventable diseases and postponable deaths are still common. Comparatively few physicians have the opportunity to practice preventive medicine, while, at the same time, physicians in general are not adequately remunerated.

Consider a survey made in two districts in the Province of Ontario, covering about five hundred physicians. These districts comprise eight counties and include a number of smaller cities.

"The average gross income, over a period of five years before 1930, was $6,262.78.

"The average cost of carrying on a practice was $2,924.46.

"The average amount of work done in a year, for which no charge was made, was $814.82.

"The average amount of work done and charged, which could not be collected, was $1,246.57."

That leaves an annual net income of $1,256.78. The depression has done little to improve these conditions.

Health or Sickness Insurance may be defined as a community organization for the prevention and cure of disease, based on contributions from those who are insured. It is a method for distributing the cost of sickness among the group. The purpose of health insurance is to facilitate the progress of medicine in its endeavour to promote health and to adjust modern medical practice to present economic conditions.

STATE HEALTH INSURANCE.

A Plan of Health Insurance for the Province of Ontario.

(From the Report of the Committee on Inter-Relations to the Ontario Medical Association, 1935.)

1. Administration.

A national plan would have many advantages over a provincial one, but social insurance legislation, of which health insurance is a part, must be introduced by the provinces. (B.N.A. Act.)

The entire project must be a business proposition between those giving and those receiving service, and should be run on a non-profit basis. Those giving the service, the professional groups concerned, shall be responsible for the quality of the service rendered. Those receiving the service, the patients, represented by departments of the provincial government, shall be responsible for the business end of the administration.

There would be complete severance of business and professional administration, though correlated under one commission, with an equal representation of those giving and those receiving service.

(a) *The commission would have the following composition:*

(1) Professional Groups:
Ontario Medical Association.

College of Physicians and Surgeons of Ontario.
Ontario Hospital Association.
Royal College of Dental Surgeons.
Ontario Nurses' Association.

(2) Patients' Groups:
Ontario Department of Health.
Ontario Department of Public Welfare.
Ontario Department of Treasury.
Ontario Manufacturers' Association.
Trades and Labor Council.

This central commission would be responsible for the complete administration of the entire project. It is believed that an autonomous, non-political commission of the above composition would assure a policy of the highest service at the lowest cost and give continuity of programme and personnel not possible under a department of government.

(b) *Officers and Committees:*

(1) Chairman and General Manager, a physician giving full-time service, appointed by the central commission. Together with the latter, he is responsible for the entire administration of the scheme, including appointment of personnel.

(2) Chief Medical Officer, responsible under the Chairman for the supervision of medical benefits and direction of Regional Medical Officers.

(3) Chief Administrative Officer, responsible under the Chairman for the supervision of business administration and Regional Administrative Officers.

(4) Professional Advisory Committees, one for each professional service provided.

(5) Lay Advisory Committees, as desired by the lay groups represented on the commission.

(c) *Regional Officers and Committees:*

(1) Regional Medical Officer, full-time physician responsible under direction of C.M.O. for the supervision of medical benefits.

(2) Regional Administrative Officer, full-time officer responsible under direction of C.A.C. for the supervision of business administration.

(4) Regional Medical Committee, composed of four, representing the local professional groups.

(5) Regional Administrative Committee, composed of three, representing the local lay groups.

The last two committees together form the Regional Health Insurance Board.

2. **Persons Included in the Plan.**

The plan, in order to carry out its principle of insurance, must be compulsory for all persons and their dependents with an income at or under a given maximum. Experience in Europe has shown that voluntary plans are successful only to the extent that indirect compulsion is exercised.

Medical care of indigents is provided for under the plan, the State paying the premiums for the indigent, who then receive medical care under the same conditions as the insured person. All the arguments in favour of having free choice of doctor, and so forth, as being conducive to good service, are equally valid for the indigent.

Persons with an income above the agreed maximum may be included on a voluntary basis for hospital benefits only.

3. **Benefits.**

It is most desirable to limit the benefits to only one, namely, medical care. It would seem that cash benefits are the root of most evils associated with European health insurance schemes.

(a) Medical Practitioner Services.

The general practitioner can give adequate and satisfactory medical care to 80 per cent. of all cases. It follows that the plan for medical benefits is based upon making available the services of the general practitioner to all.

In order that the patient may have free choice of physician, any legally qualified medical practitioner is eligible to practice under the plan, if he chooses to do so.

His duties would be:

(1) Diagnostic and Therapeutic, as far as his training and skill permit.

(2) Prophylactic:

Periodic physical examination every year available, but voluntary.

Prophylactic child care and immunization of pre-school children.

Pre-natal and post-natal care.

(b) Specialist services and all radiological and other diagnostic aids are to be secured normally through the general practitioner.

(c) Hospital care (semi-public), auxiliary services, pharmaceutical services, and visiting nursing service in the home may all be obtained through the physician, when he deems it expedient.

(d) Dental service may be arranged direct with the dentist or upon reference.

4. **Contributions.**

The guiding principles in the matter of contributions should be the spreading of costs over the entire population as well as the encouragement of early resort to medical service. Thus there should be no economic barrier imposed between doctor and patient and no bonusing for non-participation in benefits.

Obviously, the purchasing power of the low income class consumer does not permit the scheme to be self-sustaining. Contributions will, therefore, be made from the insured, the employer, the municipality, the Province, and the Federal Government.

Unit of cost is the total annual cost divided by the number of insured, exclusive of dependents.

Normal Contributions:

Insured family head....3/6 unit
Employer .....1/6 unit
Province .....1/6 unit
Federal Government ....1/6 unit

Normal Contributions of Independent Proprietors:

Insured .....4/6 unit
Province .....1/6 unit
Federal Government 1/6unit

Indigents will be classified according to income and number of dependents:

Class Insured	A At or over	B At or over	C At or over	D At or over	E Under
Single Man or Woman	600	500	400	300	300
—with 1 dependent	650	550	450	350	350
—with 2 "	700	600	500	400	400
—with 3 "	750	650	550	450	450
Proportion of premium to be paid	100%	75%	50%	25%	nil
Premium if unit cost $40, Employed	$20.00	$15.00	$10.00	$5.00	nil
Independent Proprietor	$26.66	$20.00	$13.33	$6.66	nil

Portion of indigent premiums not met by the insured:

Met by the Province.....2/3
Met by Municipality.....1/3

5. **Costs.**

The cost of medical care would be very useful to know for anybody promulgating a plan for health insurance but reliable statistics are at present lacking and future costs under a health insurance plan would doubtless be higher.

Here the consuming public must decide how much they are prepared to pay for medical service. That will determine the number and extent of benefits.

6. **Payment of Medical Practitioners.**

The Insurance Fund will be a central fund collected from all over the Province. Those areas with a small population will have to be allocated sufficient to maintain a doctor in their area. This is part of the insurance principle of spreading the cost of medical care over the province.

There are three methods of remunerating the medical practitioner: 1. *By Salary*; 2. *Per capita or panel method;* 3. *Per unit of service.*

Nearly unanimous is the opinion of the profession against the salary method, which eliminates the element of competition and thereby lowers the quality of service rendered.

Under the panel method, the physician is paid a fixed rate per capita per year. By eliminating the reporting on each individual visit, accounting and inspection costs are reduced, leaving more

money available for fees. The physician can discourage unnecessary calls at irregular hours, since his income is not immediately affected by each visit, and he can make frequent calls in acute or interesting cases without having his motive misunderstood.

The panel method also tends to promote more amicable professional relationships, since it makes it definite to which doctor a patient belongs, and permits the absence of the doctor for holidays and post-graduate study without great danger of losing his practice.

Introduced, as it was, for the medical profession in England, the panel method seems admirably adapted to their densely populated country.

In Canada, however, different conditions prevail and remuneration per unit of service would be more conducive to a high standard of service in a country so sparsely populated. This method eliminates automatically too ready reference to hospital and specialist, an outstanding weakness of the English system.

It has been recommended that the method of remuneration be uniform throughout the Province.

Criticism.

State health insurance has been and will be the subject of much controversy.

Criticisms range from fierce denunciation of such schemes as communistic, to the meek suggestion that things are really very splendid as they are now. Neither view stands close scrutiny.

On the other hand, exponents of health insurance have been carried away by their enthusiasm until they consider it as the panacea for all ills of medical practice. It must be remembered that health insurance, as social insurance in general, serves only to alleviate exigencies caused by the insufficient incomes of wage-earners in our industrialized society, and does not pretend to remove the cause of these exigencies, poverty.

The system of payment per unit of service and the greed to get "something back" would encourage a flood of patients with such minor illnesses as have no real need for medical service, This may be one cause of the rising cost of medical care under existing systems of health insurance.

In Europe, it appears that the greater the political control of the administration of health insurance, the greater is the waste and inefficiency in medical services. In Germany, 50 per cent. of the funds are swallowed by administrative bodies alone. The medical profession must be adequately represented in the control of health insurance to safeguard the interests of the public and the profession.

Another criticism of health insurance is that it does not prevent sickness. It is pointed out that, under every scheme, the amount of sickness increases, if one is to judge by the number of cases treated. To what extent this represents an increase in sickness or is due to more patients receiving the treatment required, cannot be stated.

There are many other criticisms not mentioned here, the majority of them being based on the assumption that health insurance implies an organization of medical services. Be it understood that, as far as this plan is concerned, health insurance means, not an organization of medical services, but an arrangement for the payment of these services.

The primary relations between physician and patient, as they exist to-day, will be maintained, there being no third party interposed between the patient and the doctor of his choice. This is the basis of sound medical practice.

The professional control in the administrative set-up is adequate to exclude politics from professional matters.

The system of remuneration per unit of service preserves the element of competition and individual freedom, and the fact that the physician is remunerated at all, should be conducive to a high standard of service, for in Ontario in 1932 the work paid for was only 50 per cent of the total amount of work done.

Health insurance, while not a "cure-all" and complete solution for the con-

ditions described in the introduction, is an attempt to alleviate those conditions. Its principle is that of humanitarian legislation and social insurance, which would counterbalance industrial poverty by substitution of social for personal responsibility.

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MEDICAL SOCIETY NOTES.

Under our famed and capable Al. Williams, the Medical Society Executive, in their regular weekly meetings, are planning several important social events. Bruce Tovee and his Medical-at-Home Committee have announced that the Medical-at-Home will be held at the Royal York Hotel on January 29. Irving Speigel has been elected chairman of Daffydil. To accommodate the large number of medical students and their friends, the Daffydil Committee has decided to run Daffydil three nights, March 3rd, 4th and 5th. The Graduation Banquet is being arranged by Hal. Slemon, President of Sixth Year, and will take place at the Roof Gardens, Royal York Hotel, March 12th.

Already two important entertainments, the Soph-Frost Banquet and Dance, have welcomed 4T2. Art Dobson and Bill Ortved, presidents of first and second years, are to be congratulated for these successful evenings.

A great deal of time and effort is being devoted to Open Meetings. Several topics, entertaining and educational to the members of all years, are now being arranged. As a member of the Medical Society, you cannot afford to miss these meetings.

The demand for tickets in our faculty for Hart House Musicals is overwhelming. Unfortunately, 15 to 20 per cent. of the tickets issued for each concert are not used, and the Musical Committee find it necessary, toward the end of the year, to donate part of our allotment to more-deserving colleges. May we ask your co-operation to better our reputation in this matter?

A committee, under Hugh Hayward, is being formed to consider the position of B. and M. students, and attempt to grant them membership in the Medical Society in the first years of this course.

Again this year, the Medical Society is contributing to the Bursary Fund. This will be greatly supplemented by the Board of Governors of the University.

MEDICAL SOCIETY EXECUTIVE, 1936-1937.

Hon. President—Professor W. E. Gallie, M.D., F.R.C.S. Eng., F.R.C.S. (C.).
Hon. Treasurer—Sir Frederick G. Banting, K.B.E.
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Chairman of Daffydil—I. J. Speigel, '37.
Editor of Medical Journal—H. Hayward, B.A., '37.

MEDICAL OPEN MEETINGS.

It is appropriate that the first open meeting of the Medical Society should deal with the educational problem. It is planned that the Assistant Dean, Dr. E. S. Ryerson, will give an address based upon the meeting at Atlanta in which the educational and social problems of the student were stressed. This problem is one receiving much attention in centres of learning, especially in the United States and one with which Dr. Ryerson is intimately conversant.

FEATURE SECTION

The student in Medicine must now acquire such a vast knowledge, theoretical and practical, before he begins the practice of his profession, that most of us have little time for reading or prolonged consideration of unsolved problems which we meet every day.

This column is opened in the hope that through the medium of book reviews it will help its readers to appreciate the foundations and see the direction of advance of medicine in its various fields. Also, we expect to publish the varied and interesting medical experiences of some of our fellow-students during the summer months. Such an article is that of Alistair Macdonald, which appeared in the February issue, 1936, in which he was able to discuss lead poisoning from a somewhat authoritative viewpoint. If all readers who have had a fairly intensive experience or interest in some problem would prepare an article on it, this could be made a very profitable section.

Classic Descriptions of Disease.

By Ralph H. Major,
University of Kansas School of Medicine.

The writer of this review has found that in all medical science there is considerable difficulty in mastering the essentials of a disease or other biological process if one reads only a text book description. But, if one reads the original classic description, or quotation therefrom, one often finds that understanding has now come where confusion existed. The probable reason for this is that the classic paper is written simply, clearly and with proper emphasis on the cardinal facts of the matter. On the other hand, some text books show the confusion over controversial points more clearly than these cardinal facts. In addition, a feeling of wonder and admiration sometimes strikes one upon reading an excellent description or interpretation written perhaps three hundred years ago. Such a feeling in the reader is of tremendous value in retaining the fundamentals of the particular subject.

Very many important essays are collected in this recently published book by Dr. Major, which contains 628 pages with portraits of most of the outstanding writers of medical literature. It is well worth reading from time to time, as one has occasion to look up various diseases.

J.P.F.

On the Motion of the Heart and Blood in Animals.

By William Harvey, M.D.

To appreciate the magnitude of the discovery of the circulation of the blood in 1628, one must realize the limited facilities for experimentation which Harvey possessed and the diverse views on the subject held by his contemporaries.

The heart beat had been observed but no attempt made to analyze it and although Galen had shown that the vessels contained blood alone, some learned men of the 16th century believed that the lungs sent air to the arteries which were thought to pulsate because of the respiratory movements. The evidence for such view was the usual post-mortem finding of almost empty arteries and engorged veins. Harvey deduced the reason for this finding to be because in most deaths respiration ceased before the heart beat so that blood collected in the veins for lack of the reduced intrathoracic pressure with inspiration. Numerous other examples could be quoted to show that the greatest confusion existed concerning the function of heart, blood vessels, and lungs.

Harvey was able to bring order to this chaotic state by admirable anatomical and vivisectional studies. A few of these methods might be mentioned.

He studied the anatomy of the heart and vessels in animals less complex than mammals, especially fish, which have no lungs to complicate the circulatory apparatus and in which the heart beat is much slower. In these hearts, inspection was an adequate method of study of the mechanics of the heart beat.

He estimated the rate of blood flow through various parts of human and animal bodies and thereby deduced that this blood could not all come from the food and drink imbibed, as some men believed. Thus he postulated the systemic circulation. He showed that in vivisection on snakes the clamping of the vena cava allowed the heart to empty itself of blood and cease to beat. On the other hand, clamping of the aorta caused the heart to become engorged and distended with blood and then cease to beat.

These few examples illustrate the certainty of Harvey's postulate, although he did not observe the capillaries connecting arterial and venous systems. However, one must read this 81-page thesis to appreciate the genius of the man. This thesis may be found in Camac's book, Epoch Making Contributions to Medicine, Surgery, and the Allied Sciences.

A Summer at Port Simpson, B.C.

By W. R. FEASBY, B.A., '37.

If you board the train at Toronto, say next Monday evening, you will arrive, via C.N.R. and SS. Catala from Prince Rupert, in Port Simpson early on the following Monday morning. It will probably be raining.

Port Simpson? Yes. "Port" because it is on the Pacific coast, four hundred and fifty miles north of Vancouver, one day nearer the Orient than that illustrious city, and the most westerly community in Canada; and "Simpson," after the Governor of the Hudson's Bay Company who is buried on the grounds of the post. On my arrival there, early on the 25th of May, the engineer apologized that the doctor could not meet me, as he was doing an appendectomy. And so a very full summer's experience began.

The Hospital at Port Simpson is supported by the United Church, the department of Indian affairs and the B.C. Government. It serves a community whose habits are nomadic, and who vary between three hundred and six hundred in numbers. During the summer there were about three hundred left in the village, the remainder being out fishing or at the canneries on the Skeena River some forty miles south. Consequently, the regular doctor and an assistant are kept busy on the Skeena River, while another does the work at Port Simpson. There, a thirty-five-bed hospital does every kind of work from pulling teeth to major operations. The X-Ray equipment and operating room are frequently in use and the out-patient service, including the dispensing of drugs, keeps one very busy.

During my first month on the coast I went with Dr. Perry, the chief, to the Skeena River on the hospital boat, Sunbeam III. This sturdy, thirty-five foot craft serves the canneries on the river and acts as a connecting link between the base at Port Simpson, and the branches at Port Essington and North Pacific on Skeena. In addition to general and emergency work on the river we examined some two hundred school children, and opened the hospitals for the summer work.

Perhaps the most thrilling experience of the summer was that of the night of June first. It is difficult for us inlanders to realize that in tidal river mouths, like the Skeena's, water level rises and falls over eighteen feet twice daily. This circumstance of high June tides, rapidly melting mountain snow, and torrential rain, brought about the Skeena River flood, which destroyed the railroad over three hundred miles and stopped communication by that route for six weeks. On the night to which I refer the flood was at its height and we were awakened about two a.m. to feel the boat list-

ed about thirty degrees. We had been tied to a float and scrambling out of our bunks we found that a large tree had come hurtling down under our bows and was just pulling out with considerable pounding as we arrived on deck. For half a mile across the river one could see nothing but a seething mass of drifting trees, lumber, segments of roof, stumps and all sorts of debris, hurtling by and being twisted by the treacherous eddies and swift current of the muddy brown water which flowed menacingly past with a rushing hiss to the sea. The scene was lit by a ghostly, half-clouded moon by whose light we quickly moved the boat. This was repeated twice that night, and for days we watched fearfully for the hidden snags which were floating everywhere.

Experiences during the two months at Port Simpson itself were legion. Everything from the cradle to the grave was in the doctor's care. This included the burial of one's failures. A most interesting group of cases turned up with what we have called gasoline poisoning. Gasoline engine fumes brought about temporary cessation of respirations, which was relieved by re-breathing and was followed in three days by cystitis and low-grade nephrosis. One has never seen or heard of such cases and would appreciate hearing of others' experience with their like.

The summer was most interesting and may I urge everyone, when the opportunity arises, to visit the Pacific coast. It is a land of promise and I know you will enjoy it immensely too.

Annual Meeting of Royal College of Physicians and Surgeons of Canada.

That it is to be increasingly difficult to secure hospital appointments without the fellowship from the Royal College of Physicians and Surgeons of Canada was the general feeling following the recent annual meeting of the organization held in Ottawa. Some 120 members attended from different points in the Dominion, with a representation of about 30 from Toronto.

Considerable interest centred around the fact that the University of Toronto provides instruction and guidance for this course, which has been in existence here since 1929. The entire set of examinations for the fellowship may be tried in Toronto, the primary, at the end of the third year in medicine, and the final at the completion of post-graduate work.

It was felt, therefore, that students aiming for the degree of F.R.C.S. (C.) would be well advised to try the primary examination, consisting of anatomy and physiology, at the completion of their third year, when the study of those two subjects is completed in their academic course. It was suggested also that the extra classes for further study, which run concurrently are always available and are of inestimable value to the potential candidate.

Following the conferring of honorary fellowships upon His Excellency Lord Tweedsmuir, Governor-General of Canada, introduced by Dr. Duncan Graham, immediate past president, and upon Professor Henry Asbury Christian, Professor of Physics, Harvard University, introduced by Dr. Alvah H. Gordon, vice-president in the division of medicine, fellowships in surgery were also conferred upon the only three successful candidates in the recent examinations, namely, Dr. D. E. Cannell, Dr. H. F. Robertson and Dr. H. W. Zeigler. All are graduates of the University of Toronto. Dr. James Dauphine, another Toronto graduate, received the fellowship in medicine, along with Dr. Plunkett, of Queen's Universty.

The scientific program consisted of informative papers given by Dr. Henry A. Christian on "Some Changing Views About Oedema and Diuresis;" by Dr. C. Leonard Huskins, Professor of Genetics, McGill University, on "Some Factors in the Development of Cancer in Mice," and by Dr. Paul H. Thorlakson, assistant professor of clinical surgery, University of Manitoba, on "Gastroscopy, Its Indications and Value."